1: Exp Dermatol. 1993 Dec;2(6):247-56.

Related Articles, L

Protein kinase C agonist and antagonist effects in normal human epiderm keratinocytes.

Matsui MS, Illarda I, Wang N, DeLeo VA.

Department of Dermatology, Columbia University, New York, NY 10032.

Several lines of evidence implicate protein kinase C (PKC) in the development of basal c and squamous cell carcinomas, tumors which originate from epidermal keratinocytes. To examine PKC in a model relevant to human skin, we exposed normal human epidermal keratinocytes (NHEK) in serum-free media to a variety of PKC agonists and antagonists NHEK PKC activity increased up to 10-fold within the 1st hour of exposure to tetradecanoyl phorbol acetate (TPA), and gradually returned to control values within 72 TPA-induced PKC activity was enhanced by pretreatment of cultures with protein and RNA synthesis inhibitors. TPA-induced growth arrest and differentiation was antagonize by staurosporine. Down-regulation by bryostatin pretreatment blocked TPA-stimulated differentiation. Our overall conclusion is that activation of PKC in cultured human keratinocytes is required for differentiation. These results are crucial to the analysis of compounds suspected of promoting or inhibiting epidermal tumors.

PMID: 8162345 [PubMed - indexed for MEDLINE]

1: Cancer Res. 1993 May 1;53(9):2081-6. Related Articles, L

Cell cycle arrest and growth inhibition by the protein kinase antagonist UCN-01 in human breast carcinoma cells.

Seynaeve CM, Stetler-Stevenson M, Sebers S, Kaur G, Sausville EA, Worland PJ.

Laboratory of Biological Chemistry, National Cancer Institute, National Institutes of Health, Bethesda, Maryland 20892.

UCN-01 is a derivative of staurosporine, initially developed as a potentially selective inhibitor of the Ca(2+)- and phospholipid-dependent protein kinase C, but with the capac to inhibit a number of tyrosine and serine/threonine kinases. UCN-01 inhibits the growth 5 breast carcinoma cell lines with a 50% inhibitory concentration range of 30-100 nM during 6 days of continuous exposure. In MCF-7, MDA-MB453, and SK-BR-3 cells, UCN-01 is 5-fold more potent in growth inhibition than its diastereomer UCN-02, but th compounds are equipotent in the inhibition of MDA-MB468 and H85787 cell growth. A differential sensitivity to a 24-h period of exposure to UCN-01 followed by drug remova and growth for 5 subsequent days was observed. The rank order for persistent inhibition cells by UCN-01 was MCF-7, MDA-MB453 >> SK-BR-3 > H85787 > MDA-MB468. MCF-7 and MDA-MB453 cells did not resume proliferation within the 5 days after brief exposure to UCN-01. In contrast, MDA-MB468 and H85787 cells showed no net growth inhibition after a 24-h pulse of UCN-01, followed by 5 more days of growth in drug-free medium. In MDA-MB468 cells, 150 nM UCN-01 retards but does not prevent cell cycle progression through S phase, but the cells are clearly blocked from exit of G1 and entry into S. Progression through S phase is completely inhibited by 600 nM UCN-01. The development of a G1 to S block by UCN-01 in MDA-MB468 cells occurs in conjunction with inhibition of [32P]orthophosphate labeling and decreased phosphotyrosine mass of discrete cellular phosphoproteins.

PMID: 7683251 [PubMed - indexed for MEDLINE]

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1: Biochim Biophys Acta. 1996 Dec 16;1317(3):175-82. Related Articles, L

Identification and purification of Ca2+/calmodulin-dependent protein kinase V from human gastric carcinoma.

Ohta H, Niki I, Ito T, Kato M, Nimura Y, Usuda N, Hidaka H.

Department of Pharmacology, Nagoya University School of Medicine, Japan.

We previously purified a novel Ca2+/calmodulin-dependent protein kinase (CaM kinase V, which has proven to be a member of the CaM kinase I family. Immunohistochemical staining of surgically-resected specimens from human subjects using specific antibody which reacts with CaM kinases I and V demonstrated heterogeneous distribution of CaM kinase I/V in normal gastric mucosa. The kinase was located mainly at the bottom of foveoral epithelium and in the gastric gland (< 25% immunopositive). In contrast, this kinase was abundant in various types of gastric carcinomas (> 75%), but not in gastric adenomas. Preferential and consistent presence of this kinase was confirmed by immunoblot analysis of gastric carcinoma and human gastric cancer cell lines, Kato-III a MKN-45. CaM kinase I/V was co-purified with CaM kinase II from resected gastric carcinoma using anion-exchange chromatography followed by calmodulin-affinity chromatography. The two kinases were finally separated by HPLC-based gel filtration. Purified CaM kinase I/V from gastric carcinoma did not possess detectable autophosphorylating activity, in contrast to CaM kinase II. The findings suggest CaM kinase I/V may possess abnormal biochemical properties in human gastric carcinoma, an the kinase could participate in cell growth of the carcinoma.

PMID: 8988233 [PubMed - indexed for MEDLINE]

1: Biochem Cell Biol. 1996;74(1):67-73.

Purification and characterization of the double-stranded DNA-activated protein kinase, DNA-PK, from human placenta.

Chan DW, Mody CH, Ting NS, Lees-Miller SP.

Department of Biological Sciences, University of Calgary, Canada.

The double-stranded DNA-activated protein kinase (DNA-PK) is a serine-threonine prot kinase that is composed of a large catalytic subunit (p350) and a DNA-binding protein o 70 and 80 kDa subunits known as the Ku autoantigen. When targeted to DNA by free DN ends, DNA-PK phosphorylates many DNA-binding proteins and transcription factors. Previously, DNA-PK had only been purified and characterized from transformed human tissue culture cells. Here we report that DNA-PK is an abundant protein in human placen and lymphocytes. We have purified the placental DNA-PK to homogeneity and show tha its biochemical properties are similar to those of the HeLa cell enzyme.

PMID: 9035691 [PubMed - indexed for MEDLINE]

1: J Biol Chem. 1994 Dec 2;269(48):30461-9. Related Articles, L

Purification of a serine kinase that associates with and phosphorylates human Cdc25C on serine 216.

Ogg S, Gabrielli B, Piwnica-Worms H.

Department of Physiology, Tufts University School of Medicine, Boston, Massachusetts 02111.

Human Cdc25C is a protein phosphatase that dephosphorylates and activates Cdc2-cycli B to trigger entry into mitosis. Cdc25C is itself regulated by phosphorylation. In asynchronously growing HeLa cells, we have determined that serine 216 is the major sit of Cdc25C phosphorylation. We have isolated a protein kinase that binds to Cdc25C and phosphorylates serine 216. The kinase binds within amino acids 200-256 of Cdc25C. Th region is conserved in some Cdc25 homologues and contains a putative bipartite nuclear localization signal just downstream from serine 216. Finally, the Cdc25C-associating kinase was purified over 8000-fold from rat liver as a 36-38-kDa doublet of proteins.

PMID: 7982962 [PubMed - indexed for MEDLINE]

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1: Matsui MS, Illarda I, Wang N, DeLeo VA. Related Articles, Li

Protein kinase C agonist and antagonist effects in normal human epidermal keratinocyte
Exp Dermatol. 1993 Dec;2(6):247-56.
PMID: 8162345 [PubMed - indexed for MEDLINE]

2: Qatsha KA, Rudolph C, Marme D, Schachtele C, May WS. Related Articles, Li

Go 6976, a selective inhibitor of protein kinase C, is a potent antagonist of human immunodeficiency virus 1 induction from latent/low-level-producing reservoir cells in vitro.
Proc Natl Acad Sci U S A. 1993 May 15;90(10):4674-8.
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3: Seynaeve CM, Stetler-Stevenson M, Sebers S, Kaur G, Sausville EA, Worland PJ. Related Articles, Li

Cell cycle arrest and growth inhibition by the protein kinase antagonist UCN-01 in hum breast carcinoma cells.
Cancer Res. 1993 May 1;53(9):2081-6.
PMID: 7683251 [PubMed - indexed for MEDLINE]

4: Hegemann L, Fruchtmann R, Bonnekoh B, Schmidt BH, Traber J, Mahrle G, Muller-Peddinghaus R, van Rooijen LA. Related Articles, Li

Effects of tiflucarbine as a dual protein kinase C/calmodulin antagonist on proliferation human keratinocytes and release of reactive oxygen species from human leukocytes.
Arch Dermatol Res. 1991;283(7):456-60.
PMID: 1801655 [PubMed - indexed for MEDLINE]

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1: Prostaglandins Leukot Essent Fatty Acids. 1993 May;48(5):355-61. Related Articles, L

Protein kinase A activators inhibit agonist induced prostaglandin production in human amnion.

Moore JJ, Moore RM, Collins PL.

Department of Pediatrics, Metro Health Medical Center, Cleveland, OH 44109.

Prostaglandin (PG) production by human amnion has been postulated to have a role in th onset of labor. Previous work by ourselves and others has demonstrated that oxytocin, phorbol esters and epidermal growth factor (EGF) increase PGE2 production in human amnion cells by activation of the Phospholipase C/Protein Kinase C (PKC) cascade syste The present study was undertaken to determine the effect of prior activation of the Adenylate Cyclase cascade system upon subsequent stimulation of PGE2 production by oxytocin, phorbol 12-myristate-13-acetate (PMA) or EGF in amnion cells and membrane discs. Isoproterenol, forskolin and dibutyryl cyclic adenosine monophosphate (dbcAMP) were utilized to activate the Adenylate Cyclase system at the receptor, enzyme and secon messenger level. In control amnion cells, oxytocin, PMA and EGF each provoked dose dependent increases in PGE2 production. In cells preincubated with dbcAMP, forskolin isoproterenol, agonist stimulated PGE2 production was markedly (50-90%) inhibited (p 0.01). Inhibition was dose dependent upon preincubator concentrations. Maximal inhibit by adenylate cyclase activators occurred with 2-4 h of preincubation. In membrane discs forskolin preincubation also inhibited oxytocin, PMA and EGF stimulation of PGE2 production. Activation of the Adenylate Cyclase system in human amnion cells or membrane discs inhibits the subsequent action of potent stimulators of PGE2 production human amnion.

PMID: 8391707 [PubMed - indexed for MEDLINE]

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1: J Cell Biochem. 1997 Jul 1;66(1):16-26. Related Articles, L

Heat stress induces tyrosine phosphorylation/activation of kinase FA/GSK alpha (a human carcinoma dedifferentiation modulator) in A431 cells.

Yang SD, Lee SC, Chang HC.

Department of Life Sciences, National Tsing Hua University, Hsinchu, Taiwan, R.O.C.

Exposure of A431 cells to a rapid temperature increase from 37 degrees to 46 degrees C could induce an increased expression (approximately 200% of control) and tyrosine phosphorylation/activation (approximately 300% of control) of protein kinase FA/ glycogen synthase kinase-3 alpha (kinase FA/GSK-3 alpha) in a time-dependent manner demonstrated by an anti-kinase FA/GSK-3 alpha immunoprecipitate kinase assay and by immunoblotting analysis with anti-kinase FA/GSK-3 alpha and anti-phosphotyrosine antibodies. The heat induction on the increased expression of kinase FA/GSK-3 alpha could be blocked by actinomycin D but not by genistein. In contrast, the heat induction o tyrosine phosphorylation/activation of kinase FA/GSK-3 alpha could be blocked by genistein or protein tyrosine phosphatase, indicating that heat stress induces a dual contr mechanism, namely, protein expression and subsequent tyrosine phosphorylation to caus cellular activation of kinase FA/GSK-3 alpha. Taken together, the results provide initial evidence that kinase FA/GSK-3 alpha represents a newly described heat stress-inducible protein subjected to tyrosine phosphorylation/activation, representing a new mode of sig transduction for the regulation of this human carcinoma dedifferentiation modulator and new mode of heat induction on cascade activation of a protein kinase.

PMID: 9215524 [PubMed - indexed for MEDLINE]